Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	4533	cell near9 pore	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/01/24 13:28
L2	0	l1 same electric?	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/01/24 13:28
L3	408	l1 same (electrical, potential, capacitance, resistance, conductance)	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/01/24 13:29
L4	104	l3 same surface	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/01/24 13:29
L5	84	l4 and @py<"2004"	US-PGPUB; USPAT; EPO; DERWENT	OR	OFF	2005/01/24 13:45
L6	0	("9886360").PN.	USPAT; EPO	OR	OFF	2005/01/24 13:46
L7	1	("6767515").PN.	USPAT; EPO	OR	OFF	2005/01/24 13:46

```
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TERMINAL (ENTER 1, 2, 3, OR ?):2


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 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2           "Ask CAS" for self-help around the clock
 NEWS  3  SEP 01   New pricing for the Save Answers for SciFinder Wizard within
                   STN Express with Discover!
 NEWS  4  OCT 28   KOREAPAT now available on STN
 NEWS  5  NOV 30   PHAR reloaded with additional data
 NEWS  6  DEC 01   LISA now available on STN
 NEWS  7  DEC 09   12 databases to be removed from STN on December 31, 2004
 NEWS  8  DEC 15   MEDLINE update schedule for December 2004
 NEWS  9  DEC 17   ELCOM reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  10 DEC 17   COMPUAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  11 DEC 17   SOLIDSTATE reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  12 DEC 17   CERAB reloaded; updating to resume; current-awareness
                   alerts (SDIs) affected
 NEWS  13 DEC 17   THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
 NEWS  14 DEC 30   EPFULL: New patent full text database to be available on STN
 NEWS  15 DEC 30   CAPLUS - PATENT COVERAGE EXPANDED
 NEWS  16 JAN 03   No connect-hour charges in EPFULL during January and
                   February 2005
 NEWS  17 JAN 11   CA/CAPLUS - Expanded patent coverage to include Russia
                   (Federal Institute of Industrial Property)

 NEWS EXPRESS  JANUARY 10 CURRENT WINDOWS VERSION IS V7.01a, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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FILE 'HOME' ENTERED AT 14:16:18 ON 24 JAN 2005
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```
=> file .meeting
'EVENTLINE' IS NOT A VALID FILE NAME
Enter "HELP FILE NAMES" at an arrow prompt (=>) for a list of files
that are available.  If you have requested multiple files, you can
specify a corrected file name or you can enter "IGNORE" to continue
accessing the remaining file names entered.
ENTER A FILE NAME OR (IGNORE):ignore
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                        ENTRY    SESSION
FULL ESTIMATED COST                                      0.21       0.21

FILE 'AGRICOLA' ENTERED AT 14:16:26 ON 24 JAN 2005

FILE 'BIOTECHNO' ENTERED AT 14:16:26 ON 24 JAN 2005
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=> pore and cell and electrical
L1          24 FILE AGRICOLA
L2         118 FILE BIOTECHNO
L3           0 FILE CONFSCI
L4           1 FILE HEALSAFE
L5           0 FILE IMSDRUGCONF
L6         107 FILE LIFESCI
L7           0 FILE MEDICONF
L8         250 FILE PASCAL

TOTAL FOR ALL FILES
L9         500 PORE AND CELL AND ELECTRICAL

=> l9 and (surface or top)
L10          1 FILE AGRICOLA
L11         21 FILE BIOTECHNO
L12          0 FILE CONFSCI
L13          0 FILE HEALSAFE
L14          0 FILE IMSDRUGCONF
L15         15 FILE LIFESCI
L16          0 FILE MEDICONF
L17         78 FILE PASCAL

TOTAL FOR ALL FILES
L18        115 L9 AND (SURFACE OR TOP)
```

```
=> l9 and cell attachment
L19           0 FILE AGRICOLA
L20           0 FILE BIOTECHNO
L21           0 FILE CONFSCI
L22           0 FILE HEALSAFE
L23           0 FILE IMSDRUGCONF
L24           0 FILE LIFESCI
L25           0 FILE MEDICONF
L26           0 FILE PASCAL

TOTAL FOR ALL FILES
L27           0 L9 AND CELL ATTACHMENT

=> l9 and attachment
L28           0 FILE AGRICOLA
L29           0 FILE BIOTECHNO
L30           0 FILE CONFSCI
L31           0 FILE HEALSAFE
L32           0 FILE IMSDRUGCONF
L33           0 FILE LIFESCI
L34           0 FILE MEDICONF
L35           2 FILE PASCAL

TOTAL FOR ALL FILES
L36           2 L9 AND ATTACHMENT

=> d l36 ibib abs total

L36   ANSWER 1 OF 2  PASCAL  COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on
      STN
ACCESSION NUMBER:         1997-0476832   PASCAL
COPYRIGHT NOTICE:         Copyright .COPYRGT. 1997 INIST-CNRS. All rights
                          reserved.
TITLE (IN ENGLISH):       Pulsed-laser metal contacting of biosensors on the
                          basis of crystalline enzyme-protein layer composites
AUTHOR:                   NEUBAUER A.; PENTZIEN S.; REETZ S.; KAUTEK W.; PUM D.;
                          SLEYTR U. B.
CORPORATE SOURCE:         Laboratory for Thin Film Technology, Federal Institute
                          for Materals Research and Testing, Unter den Eichen
                          87, 12205 Berlin, Germany, Federal Republic of;
                          Nanosearch Membrane Ges.m.b.H. (NSM), Hettenkofergasse
                          13/45, 1160 Vienna, Austria; Center for Ultrastructure
                          Research, Vienna University of Agriculture, Forestry
                          and Renewable Natural Resources, Gregor-Mendel-Strasse
                          33, 1180 Vienna, Austria
SOURCE:                   Sensors and actuators. B, Chemical, (1997), 40(2-3),
                          231-236, 22 refs.
                          ISSN: 0925-4005
DOCUMENT TYPE:            Journal
BIBLIOGRAPHIC LEVEL:      Analytic
COUNTRY:                  Switzerland
LANGUAGE:                 English
AVAILABILITY:             INIST-19425B, 354000068856110230
AN    1997-0476832   PASCAL
CP    Copyright .COPYRGT. 1997 INIST-CNRS. All rights reserved.
AB    Crystalline bacterial cell surface layers (S-layers) composed
      of monomolecular arrays of protein subunits are accessible to a wide
      variety of possible proteinchemical reactions. This enables the
      attachment and immobilization of enzyme molecules in a tightest
      packing, which has not been achieved with other immobilization matrices.
      When immobilized to an S-layer lattice, the enzyme entities are
      surrounded by nanometer pores. Thus, they can react
      electrochemically with the analyte liquid streaming through these
```

pores. The control over this process has to take place by way of an inert **electrical** contact in a distance of less than 1 nm. The relatively voluminous, but specially shaped sensor enzyme molecules have to be connected with an optimum metallic contact, which must not disturb the protein structure. Previously, platinum films were applied on enzyme layers immobilized on S-layer protein by argon sputtering. This conventional technique, however, exhibits substantial limitations. One, for instance, is the volume change of the S-layer/enzyme composite system when it is introduced into a conventional vacuum coating apparatus. This coating problem can be circumvented by a completely new deposition method, i.e. the pulse-laser-deposition (PLD) on protein crystal composite films with optimized laser parameters and reaction atmospheres. Enzyme activities of 70-80% were achieved, thus demonstrating that composite systems consisting of the 2D-protein-layer/enzyme/metal sequence can successfully serve as highly efficient sensor systems.

L36 ANSWER 2 OF 2 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1996-0190415 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Enhanced transport of bacteria in porous media by sediment-phase and aqueous-phase natural organic matter
AUTHOR: JOHNSON W. P.; LOGAN B. E.
CORPORATE SOURCE: Department of Geology and Geophysics, The University of Utah, Salt Lake City, UT 84112, United States
SOURCE: Water research : (Oxford), (1996), 30(4), 923-931, 3 tabl., refs. 1 p.
Illustrations; Table
ISSN: 0043-1354 CODEN: WATRAG
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-8940A, 354000044829730190

AN 1996-0190415 PASCAL
CP Copyright .COPYRGT. 1996 INIST-CNRS. All rights reserved.
AB Aqueous-phase dissolved natural organic matter (DOM) and sediment organic matter (SOM) were shown in laboratory mini-column experiments to affect the transport of bacteria within porous media. **Attachment** efficiencies of bacteria were estimated from their retention on quartz, iron oxide coated quartz (Fe-quartz), and Fe-quartz coated with SOM (SOM-Fe-quartz). Suwannee River Humic Acid (SRHA) and Soil Humic Acid (SHA) were used to represent organic matter (SOM and DOM), and were added to radiolabeled bacterial suspensions (10.sup.6 **cells**/ml, pH = 7.7) prior to transport. Coating quartz with iron oxide increased bacterial retention 160% relative to uncoated quartz. Coating Fe-quartz with SOM lowered bacterial retention, resulting in a fraction retained only 33% greater than retained on uncoated quartz. Compared to these effects, the effect of DOM on bacterial retention was secondary, and reflected the extent of DOM adsorption to the porous media. When DOM did not interact with the porous media, as in the case of quartz, bacterial retention in the presence of DOM was reduced by 20%. However, when DOM adsorption to the porous media was increased by coating the quartz with iron oxide, bacterial retention on the Fe-quartz increased by 10%. When Fe-quartz surfaces were loaded with DOM to equilibrium conditions to produce SOM-Fe-quartz, the presence of DOM in the applied solution also increased bacterial retention by 10%. The effects of DOM were the same for both types of humic acids (SHA or SRHA). These results suggest that SOM and DOM affect bacterial transport by increasing the negative surface charge of the Fe-quartz and bacteria, respectively. The largest decrease in bacterial retention (60%) was associated with coating of Fe-quartz by SOM in the absence of DOM.

```
=> 19 and potential
L37          11 FILE AGRICOLA
L38          57 FILE BIOTECHNO
L39           0 FILE CONFSCI
L40           0 FILE HEALSAFE
L41           0 FILE IMSDRUGCONF
L42          28 FILE LIFESCI
L43           0 FILE MEDICONF
L44          56 FILE PASCAL

TOTAL FOR ALL FILES
L45         152 L9 AND POTENTIAL

=> 19 and action potential
L46           0 FILE AGRICOLA
L47          10 FILE BIOTECHNO
L48           0 FILE CONFSCI
L49           0 FILE HEALSAFE
L50           0 FILE IMSDRUGCONF
L51           6 FILE LIFESCI
L52           0 FILE MEDICONF
L53           8 FILE PASCAL

TOTAL FOR ALL FILES
L54          24 L9 AND ACTION POTENTIAL

=> dup rem
ENTER L# LIST OR (END):l54
DUPLICATE IS NOT AVAILABLE IN 'IMSDRUGCONF, MEDICONF'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE
PROCESSING COMPLETED FOR L54
L55          19 DUP REM L54 (5 DUPLICATES REMOVED)

=> d l55 ibib abs total

L55   ANSWER 1 OF 19  PASCAL  COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED.
      on STN
ACCESSION NUMBER:         2004-0298608   PASCAL
COPYRIGHT NOTICE:         Copyright .COPYRGT. 2004 INIST-CNRS. All rights
                          reserved.
TITLE (IN ENGLISH):       Suppression of electrical alternans by
                          overexpression of HERG in canine ventricular myocytes
AUTHOR:                   FEI HUA; JOHNS David C.; GILMOUR Robert F. JR
CORPORATE SOURCE:         Department of Biomedical Sciences, Cornell University,
                          Ithaca, New York 14853, United States; Department of
                          Neurosurgery, Johns Hopkins School of Medicine,
                          Baltimore, Maryland 21205, United States
SOURCE:                   American journal of physiology. Heart and circulatory
                          physiology, (2004), 55(6), 2342-2352, 39 refs.
                          ISSN: 0363-6135  CODEN: AJPPDI
DOCUMENT TYPE:            Journal
BIBLIOGRAPHIC LEVEL:      Analytic
COUNTRY:                  United States
LANGUAGE:                 English
AVAILABILITY:             INIST-670D, 354000111978950380
AN    2004-0298608   PASCAL
CP    Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
AB    Suppression of electrical alternans may be antiarrhythmic. Our
      previous computer simulations have suggested that increasing the rapid
      component of the delayed rectifier K.sup.+ current (I.sub.K.sub.r)
      suppresses alternans. To test this hypothesis, I.sub.K.sub.r in isolated
      canine ventricular myocytes was increased by infection with an adenovirus
```

containing the gene for the **pore**-forming domain of I.sub.K.sub.r [human ether-a-go-go gene (HERG)]. With the use of the perforated or whole **cell** patch-clamp technique, **action potentials** recorded at different pacing cycle lengths (CLs) were applied to the myocytes as the command waveforms. HERG infection markedly increased peak I.sub.K.sub.r during the **action potential** (from 0.54 ± 0.03 pA/pF in control to 3.60 ± 0.81 pA/pF). Rate-dependent alterations of peak I.sub.K.sub.r were similar for freshly isolated myocytes and HERG-infected myocytes. In both **cell** types, I.sub.K.sub.r increased when CL decreased from 1,000 to 500 ms and then decreased progressively as CL decreased further. During alternans at CL = 170 ms, peak I.sub.K.sub.r was larger for the short than for the long **action potential** for both groups, but the difference in peak I.sub.K.sub.r was larger for HERG-infected myocytes. The voltage at which peak I.sub.K.sub.r occurred was significantly less negative in HERG-infected myocytes, in association with shifts of the steady-state voltage-dependent activation and inactivation curves to less negative potentials. Pacing at short CL induced stable alternans in freshly isolated myocytes and in cultured myocytes without HERG infection, but not in HERG-infected myocytes. These data support the idea that increasing I.sub.K.sub.r may be a viable approach to suppressing **electrical** alternans.

L55 ANSWER 2 OF 19 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2004-0594545 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Heart rate lowering by specific and selective If current inhibition with ivabradine: A new therapeutic perspective in cardiovascular disease
AUTHOR: DIFRANCESCO Dario; CAMM John A.
CORPORATE SOURCE: Dipartimento di Scienze Biomolecolari e Biotecnologie, Universita di Milano, Milan, Italy; The Medical School, St George's Hospital, London, United Kingdom
SOURCE: Drugs : (Basel), (2004), 64(16), 1757-1765, 66 refs. ISSN: 0012-6667 CODEN: DRUGAY
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: New Zealand
LANGUAGE: English
AVAILABILITY: INIST-15326, 354000122211320030

AN 2004-0594545 PASCAL
CP Copyright .COPYRGT. 2004 INIST-CNRS. All rights reserved.
AB Resting heart rate is associated with cardiovascular and all-cause mortality, and the mortality benefit of some cardiovascular drugs seems to be related in part to their heart rate-lowering effects. Since it is difficult to separate the benefit of heart rate lowering from other actions with currently available drugs, a 'pure' heart rate-lowering drug would be of great interest in establishing the benefit of heart rate reduction per se. Heart rate is determined by spontaneous **electrical** pacemaker activity in the sinoatrial node. Cardiac pacemaker **cells** generate the spontaneous slow diastolic depolarisation that drives the membrane voltage away from a hyperpolarised level towards the threshold level for initiating a subsequent **action potential**, generating rhythmic **action potentials** that propagate through the heart and trigger myocardial contraction. The If current is an ionic current that determines the slope of the diastolic depolarisation, which in turn controls the heart beating rate. Ivabradine is the first specific heart rate-lowering agent to have completed clinical development for stable angina pectoris. Ivabradine specifically blocks cardiac pacemaker **cell** f-channels by entering and binding to a site in the channel **pore** from the intracellular side. Ivabradine is selective for the

I.sub.f current and exerts significant inhibition of this current and heart rate reduction at concentrations that do not affect other cardiac ionic currents. This activity translates into specific heart rate reduction, which reduces myocardial oxygen demand and simultaneously improves oxygen supply, by prolonging diastole and thus allowing increased coronary flow and myocardial perfusion. Ivabradine lowers heart rate without any negative inotropic or lusitropic effect, thus preserving ventricular contractility. Ivabradine was shown to reduce resting heart rate without modifying any major electrophysiological parameters not related to heart rate. In patients with left ventricular dysfunction, ivabradine reduced resting heart rate without altering myocardial contractility. Thus, pure heart rate lowering can be achieved in the clinic as a result of specific and selective I.sub.f current inhibition. Two randomised clinical studies have shown that ivabradine is an effective anti-ischaemic agent that reduces heart rate and improves exercise capacity in patients with stable angina. Ivabradine was shown to be superior to placebo in improving exercise tolerance test (ETT) criteria (n = 360) and, in a 4-month, double-blind, controlled study (n = 939), ivabradine 5 and 7.5mg twice daily were shown to be at least as effective as atenolol 50 and 100mg once daily, respectively, in improving total exercise duration and other ETT criteria, and reducing the number of angina attacks. Experimental data indicate a potential role of pure heart rate lowering in other cardiovascular conditions, such as heart failure.

L55 ANSWER 3 OF 19 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2005-0016624 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Transgenic upregulation of I.sub.K.sub.1 in the- mouse heart leads to multiple abnormalities of cardiac excitability
AUTHOR: JINGDONG LI; MCLERIE Meredith; LOPATIN Anatoli N.
CORPORATE SOURCE: Department of Molecular and Integrative Physiology, University of Michigan Medical School, Ann Arbor, Michigan 48109, United States
SOURCE: American journal of physiology. Heart and circulatory physiology, (2004), 56(6), H2790-H2802, 42 refs. ISSN: 0363-6135 CODEN: AJPPDI
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670D, 354000122548360520

AN 2005-0016624 PASCAL
CP Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.
AB To assess the functional significanc of upregulation of the cardiac current (I.sub.K.sub.1), we have produced anc characterized the first transgenic (TG) mouse model of I.sub.K.sub.1 upregula tion. To increase I.sub.K.sub.1 density, a **pore**-forming subunit of the Kir2.1 (green fluorescent protein-tagged) channel was expressed in the hear under control of the α-myosin heavy chain promoter. Two lines of TC animals were established with a high level of TG expression in al major parts of the heart: line 1 mice were characterized by 14% hear hypertrophy and a normal life span; line 2 mice displayed an increase mortality rate, and in mice <=1 mo old, heart weight-to-body weigh ratio was increased by >100%. In adult ventricular myocytes expressing the Kir2.1-GFP subunit, I.sub.K.sub.1 conductance at the reversal potentia was increased .eqvsim.9- and .eqvsim.10-fold in lines I and 2, respectively Expression of the Kir2. transgene in line 2 ventricular myocytes was heterogeneous when assayed by single-**cell** analysis of GFP fluores cence. Surface ECG recordings in line 2 mice revealed numerous abnormalities of excitability, including slowed heart rate,

premature ventricular contractions, atrioventricular block, and atrial fibrillation Line 1 mice displayed a less severe phenotype. In both TG lines **action potential** duration at 90% repolarization and monophasic actior potential at 75-90% repolarization were significantly reduced, leading to neuronlike **action potentials**, and the slow phase of the T wave way abolished, leading to a short Q-T interval. This study provides a new TG model of I.sub.K.sub.1 upregulation, confirms the significant role of I.sub.K.sub.1ir cardiac excitability, and is consistent with adverse effects of I.sub.K upregulation on cardiac **electrical** activity.

L55 ANSWER 4 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2003:36183358 BIOTECHNO
TITLE: It takes two to tango, but three to I.sub.S.sub.A
AUTHOR: Herson P.S.; Adelman J.P.
CORPORATE SOURCE: P.S. Herson, Vollum Institute, Oregon Hlth. and Sciences University, 3181 S.W. Sam Jackson Park Road, Portland, OR 97239, United States.
SOURCE: Neuron, (06 FEB 2003), 37/3 (370-372), 6 reference(s)
CODEN: NERNET ISSN: 0896-6273
DOCUMENT TYPE: Journal; (Short Survey)
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2003:36183358 BIOTECHNO
AB Rapidly inactivating A-type potassium channels are important determinants of firing frequency in many excitable **cells**. Nadal et al. (in this issue of Neuron) purified A-type potassium (I.sub.S.sub.A) channels from rat cerebellum and identified a novel β subunit. This protein, DPPX, associates with the **pore**-forming subunits and endows previously elusive kinetic properties on A-type channels formed from cloned subunits.

L55 ANSWER 5 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2002:34252231 BIOTECHNO
TITLE: An unexpected role for brain-type sodium channels in coupling of **cell** surface depolarization to contraction in the heart
AUTHOR: Maier S.K.G.; Westenbroek R.E.; Schenkman K.A.; Feigl E.O.; Scheuer T.; Catterall W.A.
CORPORATE SOURCE: W.A. Catterall, University of Washington, Department of Pharmacology, Campus Box 357280, 1959 NE Pacific Street, Seattle, WA 98195, United States.
E-mail: wcatt@u.washington.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (19 MAR 2002), 99/6 (4073-4078), 33 reference(s)
CODEN: PNASA6 ISSN: 0027-8424
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2002:34252231 BIOTECHNO
AB Voltage-gated sodium channels composed of **pore**-forming α and auxiliary β subunits are responsible for the rising phase of the **action potential** in cardiac muscle, but the functional roles of distinct sodium channel subtypes have not been clearly defined. Immunocytochemical studies show that the principal cardiac poreforming α subunit isoform Na.sub.v1.5 is preferentially localized in intercalated disks, whereas the brain α subunit isoforms Na.sub.v1.1, Na.sub.v1.3, and Na.sub.v1.6 are localized in the transverse tubules. Sodium currents due to the highly tetrodotoxin (TTX)-sensitive brain isoforms in the transverse tubules are small and are detectable only after activation with β scorpion toxin. Nevertheless, they play

an important role in coupling depolarization of the **cell** surface membrane to contraction, because low TTX concentrations reduce left ventricular function. Our results suggest that the principal cardiac isoform in the intercalated disks is primarily responsible for **action potential** conduction between **cells** and reveal an unexpected role for brain sodium channel isoforms in the transverse tubules in coupling **electrical** excitation to contraction in cardiac muscle.

L55 ANSWER 6 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2002:34625487 BIOTECHNO
TITLE: New perspectives on the structure and function of the Na.sup.+ channel multigene family
AUTHOR: Ogata N.; Yoshida S.
CORPORATE SOURCE: N. Ogata, Department of Physiology, Hiroshima Univ. Sch. of Medicine, Hiroshima 734-8551, Japan. E-mail: ogatan@hiroshima-u.ac.jp
SOURCE: Current Medicinal Chemistry - Central Nervous System Agents, (2002), 2/1 (59-81), 108 reference(s) CODEN: CMCCCO ISSN: 1568-0150
DOCUMENT TYPE: Journal; General Review
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2002:34625487 BIOTECHNO
AB Recent studies on the voltage-gated Na.sup.+ channel (VGSC) have revealed several excellent discoveries regarding its structure and function. This article summarizes recent findings on VGSCs, and presents our views on the subject. Based on the multi-**pore** 3D model of the VGSC, we propose a "twist-sprinkler" model: (i) twisting and untwisting of the central cavity corresponds to the closed and open states of the channel, and (ii) cytoplasmic outlet **pores** sprinkle Na.sup.+ ions laterally over the inner surface of the plasma membrane to effect a rapid depolarization. VGSCs can be classified into two major categories. Category-I isoforms currently comprise nine highly homologous clones (Na.sub.v 1.1- Na.sub.v 1.9), most of which have been functionally expressed. In contrast, the category-II isoform consists of one clone (Na.sub.x), which has not been successfully expressed in an exogenous system. It is considerably different from the category-I isoforms, especially in the S4 segment, and shows little voltage dependence. The main function of the category-I isoforms is to form an **action potential** upstroke. However, Na.sub.v 1.6 can also influence subthreshold **electrical** activity in neurons through the "persistent" and "resurgent" Na.sup.+ currents, indicating that the VGSC itself can modulate overall neuronal firing behavior. Na.sub.v 1.8 and Na.sub.v 1.9 are preferentially expressed in peripheral nociceptive neurons and contain a structure common to tetrodotoxin (TTX)-resistant Na.sup.+ channels. Both Na.sub.v 1.8 and Na.sub.v 1.9 play a pivotal role in pain sensation. The category-II isoform Na.sub.x (x = unknown function) is a "concentration-sensitive" but not "voltage-sensitive" Na.sup.+ channel. It is involved in regulation of salt intake behavior by sensing an increase in [Na.sup.+]o, and it should be renamed as Na.sub.c (c = concentration).

L55 ANSWER 7 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 2002:35174982 BIOTECHNO
TITLE: Specific contribution of human T-type calcium channel isotypes (α.sub.1.sub.G, α.sub.1.sub.H and α.sub.1.sub.I) to neuronal excitability
AUTHOR: Chemin J.; Monteil A.; Perez-Reyes E.; Bourinet E.; Nargeot J.; Lory P.
CORPORATE SOURCE: P. Lory, Inst. de Genetiq. Humaine, CNRS UPR 1142, 141 rue de la Cardonille, F-34396 Montpellier Cedex 05,

France.
E-mail: philippe.lory@igh.cnrs.fr
SOURCE: Journal of Physiology, (01 APR 2002), 540/1 (3-14), 51 reference(s)
CODEN: JPHYA7 ISSN: 0022-3751
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2002:35174982 BIOTECHNO
AB In several types of neurons, firing is an intrinsic property produced by specific classes of ion channels. Low-voltage-activated T-type calcium channels (T-channels), which activate with small membrane depolarizations, can generate burst firing and pacemaker activity. Here we have investigated the specific contribution to neuronal excitability of cloned human T-channel subunits. Using HEK-293 **cells** transiently transfected with the human α.sub.1.sub.G (Ca.sub.v3.1), α.sub.1.sub.H (Ca.sub.v3.2) and α.sub.1.sub.I (Ca.sub.v3.3) subunits, we describe significant differences among these isotypes in their biophysical properties, which are highlighted in **action potential** clamp studies. Firing activities occurring in cerebellar Purkinje neurons and in thalamocortical relay neurons used as voltage clamp waveforms revealed that α.sub.1.sub.G channels and, to a lesser extent, α.sub.1.sub.H channels produced large and transient currents, while currents related to α.sub.1.sub.G channels exhibited facilitation and produced a sustained calcium entry associated with the depolarizing after-potential interval. Using simulations of reticular and relay thalamic neuron activities, we show that α.sub.1.sub.I currents contributed to sustained **electrical** activities, while α.sub.1.sub.G and α.sub.1.sub.H currents generated short burst firing. Modelling experiments with the NEURON model further revealed that the α.sub.1.sub.G channel and α.sub.1.sub.I channel parameters best accounted for T-channel activities described in thalamocortical relay neurons and in reticular neurons, respectively. Altogether, the data provide evidence for a role of α.sub.1.sub.I channel in pacemaker activity and further demonstrate that each T-channel **pore**-forming subunit displays specific gating properties that account for its unique contribution to neuronal firing.

L55 ANSWER 8 OF 19 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002-0416439 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2002 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Block of the background K.sup.+ channel TASK-1 contributes to arrhythmogenic effects of platelet-activating factor
AUTHOR: BARBUTI Andrea; ISHII Satoshi; SHIMIZU Takao; ROBINSON Richard B.; FEINMARK Steven J.
CORPORATE SOURCE: Center for Molecular Therapeutics, Department of Pharmacology, Columbia University, New York, New York 10032, United States; Department of Biochemistry and Molecular Biology, University of Tokyo, Tokyo, 113-003, Japan
SOURCE: American journal of physiology. Heart and circulatory physiology, (2002), 51(6), H2024-H2030, 26 refs.
ISSN: 0363-6135 CODEN: AJPPDI
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-670D, 354000100717110100
AN 2002-0416439 PASCAL

AB Platelet-activating factor (PAF), an inflammatory phospholipid, induces ventricular arrhythmia via an unknown ionic mechanism. We can now link PAF-mediated cardiac electrophysiological effects to inhibition of a two-**pore** domain K.sup.+ channel [TWIK-related acid-sensitive K.sup.+ background channel (TASK-1)]. Superfusion of carbamyl-PAF (C-PAF), a stable analog of PAF, over murine ventricular myocytes causes abnormal automaticity, plateau phase arrest of the **action potential**, and early afterdepolarizations in paced and quiescent **cells** from wild-type but not PAF receptor knockout mice. C-PAF-dependent currents are insensitive to Cs.sup.+ and are outwardly rectifying with biophysical properties consistent with a K.sup.+-selective channel. The current is blocked by TASK-1 inhibitors, including protons, Ba.sup.2.sup.+, Zn.sup.2.sup.+, and methanandamide, a stable analog of the endogenous lipid ligand of cannabanoid receptors. In addition, when TASK-1 is expressed in CHO **cells** that express an endogenous PAF receptor, superfusion of C-PAF decreases the expressed current. Like C-PAF, methanandamide evoked spontaneous activity in quiescent myocytes. C-PAF- and methanandamide-sensitive currents are blocked by a specific protein kinase C (PKC) inhibitor, implying overlapping signaling pathways. In conclusion, C-PAF blocks TASK-1 or a closely related channel, the effect is PKC dependent, and the inhibition alters the **electrical** activity of myocytes in ways that would be arrhythmogenic in the intact heart.

L55 ANSWER 9 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2001:34073220 BIOTECHNO
TITLE: Electroporation in a model of cardiac defibrillation
AUTHOR: Ashihara T.; Yao T.; Namba T.; Ito M.; Ikeda T.; Kawase A.; Toda S.; Suzuki T.; Inagaki A.; Sugimachi M.; Kinoshita M.; Nakazawa K.
CORPORATE SOURCE: Dr. T. Ashihara, First Dept. of Internal Medicine, Shiga University of Medical Science, Seta Tsukinowa-cho, Otsu-city 520-2192, Japan. E-mail: ash@belle.shiga-med.ac.jp
SOURCE: Journal of Cardiovascular Electrophysiology, (2001), 12/12 (1393-1403), 47 reference(s) CODEN: JCELE2 ISSN: 1045-3873
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 2001:34073220 BIOTECHNO
AB Introduction: It is known that high-strength shock disrupts the lipid matrix of the myocardial **cell** membrane and forms reversible aqueous **pores** across the membrane. This process is known as "electroporation." However, it remains unclear whether electroporation contributes to the mechanism of ventricular defibrillation. The aim of this computer simulation study was to examine the possible role of electroporation in the success of defibrillation shock. Methods and Results: Using a modified Luo-Rudy-1 model, we simulated two-dimensional myocardial tissue with a homogeneous bidomain nature and unequal anisotropy ratios. Spiral waves were induced by the S1-S2 method. Next, monophasic defibrillation shocks were delivered externally via two line electrodes. For nonelectroporating tissue, termination of ongoing fibrillation succeeded; however, new spiral waves were initiated, even with high-strength shock (24 V/cm). For electroporating tissue, high-strength shock (24 V/cm) was sufficient to extinguish ongoing fibrillation and did not initiate any new spiral waves. Weak shock (16 to 20 V/cm) also extinguished ongoing fibrillation; however, in contrast to the high-strength shock, new spiral waves were initiated. Success in defibrillation depended on the occurrence of electroporation-mediated anodal-break excitation from the physical anode and the virtual anode. Some excitation wavefronts following **electrical** shock used a

deexcited area with recovered excitability as a pass-through point; therefore, electroporation-mediated anodal-break excitation is necessary to block out the pass-through point, resulting in successful defibrillation. Conclusion: The electroporation-mediated anodal-break excitation mechanism may play an important role in **electrical** defibrillation.

L55 ANSWER 10 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE

ACCESSION NUMBER: 1999:29243164 BIOTECHNO
TITLE: Structure and function of cardiac potassium channels
AUTHOR: Snyders D.J.
CORPORATE SOURCE: D.J. Snyders, Department of Molecular Biophysics, Department of Biochemistry (UIA), University of Antwerp, Universiteitsplein 1 - T4.21, B-2610 Antwerp, Belgium.
E-mail: dsnyders@uia.ua.ac.be
SOURCE: Cardiovascular Research, (1999), 42/2 (377-390), 114 reference(s)
CODEN: CVREAU ISSN: 0008-6363
PUBLISHER ITEM IDENT.: S0008636399000711
DOCUMENT TYPE: Journal; General Review
COUNTRY: Netherlands
LANGUAGE: English
SUMMARY LANGUAGE: English

AN 1999:29243164 BIOTECHNO
AB Recent advances in molecular biology have had a major impact on our understanding of the biophysical and molecular properties of ion channels. This review is focused on cardiac potassium channels which, in general, serve to control and limit cardiac excitability. Approximately 60 K.sup.+ channel subunits have been cloned to date. The (evolutionary) oldest potassium channel subunits consist of two transmembrane (Tm) segments with an intervening **pore**-loop (P). Channels formed by four 2Tm-1P subunits generally function as inwardly rectifying K.sup.+-selective channels (KirX.Y): they conduct substantial current near the resting potential but carry little or no current at depolarized potentials. The inward rectifier I(K1) and the ligand-gated K(ATP) and K(ACh) channels are composed of such subunits. The second major class of K.sup.+ channel subunits consists of six transmembrane segments (S1- S6). The S5-P-S6 section resembles the 2Tm-1P subunit, and the additional membrane-spanning segments (especially the charged S4 segment) endow these 6Tm-1P channels with voltage-dependent gating. For both major families, four subunits assemble into a homo- or heterotetrameric channel, subject to specific subunit-subunit interactions. The 6Tm-1P channels are closed at the resting potential, but activate at different rates upon depolarization to carry sustained or transient outward currents (the latter due to inactivation by different mechanisms). Cardiac **cells** typically display at least one transient outward current and several delayed rectifiers to control the duration of the **action potential**. The molecular basis for each of these currents is formed by subunits that belong to different Kvx.y subfamilies and alternative splicing can contribute further to the diversity in native **cells**. These subunits display distinct pharmacological properties and drug-binding sites have been identified. Additional subunits have evolved by concatenation of two 2Tm-1P subunits (4Tm-2P); dimers of such subunits yield voltage- independent leak channels. A special class of 6Tm-1P subunits encodes the 'funny' pacemaker current which activates upon hyperpolarization and carries both Na.sup.+ and K.sup.+ ions. The regional heterogeneity of K.sup.+ currents and **action potential** duration is explained by the heterogeneity of subunit expression, and significant changes in expression occur in cardiac disease, most frequently a reduction. This **electrical** remodelling may also be important for novel antiarrhythmic therapeutic strategies. The recent crystallization of a

2Tm-1P channel enhances the outlook for more refined molecular approaches.

L55 ANSWER 11 OF 19 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1998-0450199 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 1998 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Functional knockout of the transient outward current, long-QT syndrome, and cardiac remodeling in mice expressing a dominant-negative Kv4 α subunit
AUTHOR: BARRY D. M.; HAODONG XU; SCHUESSLER R. B.; NERBONNE J. M.
CORPORATE SOURCE: Department of Molecular Biology and Pharmacology, Washington University Medical School, St Louis, Mo, United States; Department of Surgery, Washington University Medical School, St Louis, Mo, United States
SOURCE: Circulation research, (1998), 83(5), 560-567, 34 refs. ISSN: 0009-7330 CODEN: CIRUAL
DOCUMENT TYPE: Journal; Short communication
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-7216, 354000070299530110
AN 1998-0450199 PASCAL
CP Copyright .COPYRGT. 1998 INIST-CNRS. All rights reserved.
AB A novel in vivo experimental strategy, involving **cell** type-specific expression of a dominant-negative K.sup.+ channel **pore**-forming α subunit, was developed and exploited to probe the molecular identity of the cardiac transient outward K.sup.+ current (I.sub.t.sub.o). A point mutation (W to F) was introduced at position 362 in the **pore** region of Kv4.2 to produce a nonconducting mutant (Kv4.2W362F) subunit. Coexpression of Kv4.2W362F with Kv4.2 (or Kv4.3) attenuates the wild-type currents, and the effect is subfamily specific; ie, Kv4.2W362F does not affect heterologously expressed Kv1.4 currents. With the use of the α-myosin heavy chain promoter to direct cardiac-specific expression, several lines of Kv4.2W362F transgenic mice were generated. Electrophysiological recordings reveal that I.sub.t.sub.o is selectively eliminated in ventricular myocytes isolated from transgenic mice expressing Kv4.2W362F, thereby demonstrating directly that the Kv 4 subfamily underlies I.sub.t.sub.o in the mammalian heart. Functional knockout of I.sub.t.sub.o leads to marked increases in **action potential** durations in ventricular myocytes and to prolongation of the QT interval in surface ECG recordings. In addition, a novel rapidly activating and inactivating K.sup.+ current, which is not detectable in myocytes from nontransgenic littermates, is evident in Kv4.2W362F-expressing ventricular **cells**. Importantly, these results demonstrate that **electrical** remodeling occurs in the heart when the expression of endogenous K.sup.+ channels is altered.

L55 ANSWER 12 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN

ACCESSION NUMBER: 1997:27289761 BIOTECHNO
TITLE: Molecular mechanism and functional significance of the MinK control of the KvLQT1 channel activity
AUTHOR: Romey G.; Attali B.; Chouabe C.; Abitbol I.; Guillemare E.; Barhanin J.; Lazdunski M.
CORPORATE SOURCE: M. Lazdunski, Inst. Pharmacol. Molec./Cellulaire, CNRS, 660 route des Lucioles, Sophia Antipolis, 06560 Valbonne, France.
E-mail: ipmc@unice.fr
SOURCE: Journal of Biological Chemistry, (1997), 272/27 (16713-16716), 28 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258

DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1997:27289761 BIOTECHNO
AB The very slowly activating delayed rectifier K.sup.- channel I(Ks) is essential for controlling the repolarization phase of cardiac **action potentials** and K.sup.- homeostasis in the inner ear. The I(Ks) channel is formed via the assembly of two transmembrane proteins, KvLQT1 and MinK. Mutations in KvLQT1 are associated with a long QT syndrome that causes syncope and sudden death and also with deafness. Here, we show a new mode of association between ion channel forming subunits in that the cytoplasmic C-terminal end of Mink interacts directly with the **pore** region of KvLQT1. This interaction reduces KvLQT1 channel conductance from 7.6 to 0.58 picosiemens. However, because Mink also reveals a large number of previously silent KvLQT1 channels (x 60), the overall effect is a large increase (x 4) in the macroscopic K.sup.- current. Conformational changes associated with the KvLQT1/MinK association create very slow and complex activation kinetics without much alteration in the deactivation process. Changes induced by Mink have an essential regulatory role in the development of this K.sup.- channel activity upon repetitive **electrical** stimulation with a particular interest in tachycardia.

L55 ANSWER 13 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE
ACCESSION NUMBER: 1996:26424228 BIOTECHNO
TITLE: Absence of the β subunit (cchb1) of the skeletal muscle dihydropyridine receptor alters expression of the α.sub.1 subunit and eliminates excitation-contraction coupling
AUTHOR: Gregg R.G.; Messing A.; Strube C.; Beurg M.; Moss R.; Behan M.; Sukhareva M.; Haynes S.; Powell J.A.; Coronado R.; Powers P.A.
CORPORATE SOURCE: R.G. Gregg, Waisman Center, University of Wisconsin, 1500 Highland Avenue, Madison, WI 53705, United States.
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1996), 93/24 (13961-13966) CODEN: PNASA6 ISSN: 0027-8424
DOCUMENT TYPE: Journal; Conference Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1996:26424228 BIOTECHNO
AB The multisubunit (α(1S), α.sub.2/δ, β.sub.1, and γ) skeletal muscle dihydropyridine receptor transduces transverse tubule membrane depolarization into release of Ca.sup.2.sup.+ from the sarcoplasmic reticulum, and also acts as an L-type Ca.sup.2.sup.+ channel. The α(1S) subunit contains the voltage sensor and channel **pore**, the kinetics of which are modified by the other subunits. To determine the role of the β.sub.1 subunit in channel activity and excitation-contraction coupling we have used gene targeting to inactivate the β.sub.1 gene. β.sub.1-null mice die at birth from asphyxia. **Electrical** stimulation of β.sub.1-null muscle fails to induce twitches, however, contractures are induced by caffeine. In isolated β.sub.1-null myotubes, **action potentials** are normal, but fail to elicit a Ca.sup.2.sup.+ transient. L-type Ca.sup.2.sup.+ current is decreased 10- to 20-fold in the β.sub.1-null **cells** compared with littermate controls. Immunohistochemistry of cultured myotubes shows that not only is the β.sub.1 subunit absent, but the amount of α(1S) in the membrane also is undetectable. In contrast, the β.sub.1 subunit is localized appropriately in dysgenic, mdg/mdg, (α(1S)-null)

cells. Therefore, the β.sub.1 subunit may not only play an important role in the transport/insertion of the α(1S) subunit into the membrane, but may be vital for the targeting of the muscle dihydropyridine receptor complex to the transverse tubule/sarcoplasmic reticulum junction.

L55 ANSWER 14 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN
DUPLICATE
ACCESSION NUMBER: 1996:26266146 BIOTECHNO
TITLE: Class III antiarrhythmic effects of zatebradine: Time-, state-, use-, and voltage-dependent block of hKv1.5 channels
AUTHOR: Valenzuela C.; Delpon E.; Franqueza L.; Gay P.; Perez O.; Tamargo J.; Snyders D.J.
CORPORATE SOURCE: Inst. of Pharmacology and Toxicology, School of Medicine, Universidad Complutense,28040 Madrid, Spain.
SOURCE: Circulation, (1996), 94/3 (562-570)
CODEN: CIRCAZ ISSN: 0009-7322
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1996:26266146 BIOTECHNO
AB Background: Zatebradine is a bradycardic agent that inhibits the hyperpolarization-activated current (I(f)) in the rabbit sinoatrial node. It also prolongs **action potential** duration in papillary muscles in guinea pigs and in Purkinje fibers in rabbits. The underlying mechanism by which zatebradine induces this effect has not been explored, but it is likely to involve K.sup.+ channel block. Methods and Results: Cloned human cardiac K.sup.+ delayed rectifier currents (hKv1.5) were recorded in Ltk **cells** transfected with their coding sequence. Zatebradine 10 μmol/L did not modify the initial activation time course of the current but induced a subsequent decline to a lower steady-state current level with a time constant of 109±16 ms. Zatebradine inhibited hKv1.5 with an apparent K(D) of 1.86±0.14 μmol/L. Block was voltage dependent (**electrical** distance δ=0.177±0.003) and accumulated in a use-dependent manner during 0.5- and 1-Hz pulse trains because of slower recovery kinetics in the presence of the drug. Zatebradine reduced the tail current amplitude, recorded at -30 mV, and slowed the deactivation time course, which resulted in a 'crossover' phenomenon when control and zatebradine tail currents were superimposed. Conclusions: These results indicate that (1) zatebradine is an open-channel blocker of hKv1.5, (2) binding occurs in the internal mouth of the ion **pore**, (3) unbinding is required before the channel can close, and (4) zatebradine-induced block is use dependent because of slower recovery kinetics in the presence of the drug. These effects may explain the prolongation of the cardiac **action potential** and could be clinically relevant.

L55 ANSWER 15 OF 19 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 95:116217 LIFESCI
TITLE: Surfing the DNA databases for K super(+) channels nets yet more diversity
AUTHOR: Salkoff, L.; Jegla, T.
CORPORATE SOURCE: Dep. Anat. and Neurobiol., Washington Univ. Sch. Med., St. Louis, MO 63110, USA
SOURCE: NEURON, (1995) vol. 15, no. 3, pp. 489-493.
ISSN: 0896-6273.
DOCUMENT TYPE: Journal
FILE SEGMENT: N3
LANGUAGE: English
AB K super(+) channels are a life and death matter. Perhaps the best assessment of whether a **cell** is living or dead is whether or not it has a membrane potential, and K super(+) channels have a significant

role in setting the membrane potential in **cells** from a wide variety of life forms. In addition to this life or death matter, K super(+) channels serve a host of other functions relating to the **electrical** lives of **cells**, like setting the frequency and duration of **action potentials** and, in general, shaping the **electrical** activity of **cells**. Because multiple K super(+) channel types have also been found in a wide variety of **cells** that are not known to be electrically excitable, it is likely that we don't yet comprehend all of the functions of these versatile proteins. Data now surfacing from the various genomic DNA sequencing projects suggest that this family of proteins might be even more diverse than previously imagined. The TOK1 channel is novel, not only because of its unique subunit structure (it resembles two K super(+) channel subunits of different classes linked together) and its unique physiology (it is outwardly rectifying by a nonconventional mechanism), but also because of the way in which this unique channel was found. Rather than using the conventional experimental methods of molecular biology in a wet lab, the authors discovered TOK1 by surfing the public DNA database for one of the rare and special ancient conserved protein regions that have been identified as a result of the genome sequencing projects. This particular ancient conserved region is the universal signature of the ion-selective **pore** of K super(+) channels.

L55 ANSWER 16 OF 19 BIOTECHNO COPYRIGHT 2005 Elsevier Science B.V. on STN DUPLICATE

ACCESSION NUMBER: 1993:23197520 BIOTECHNO
TITLE: The cloning and expression of a sodium channel β1-subunit cDNA from human brain
AUTHOR: McClatchey A.I.; Cannon S.C.; Slaugenhaupt S.A.; Gusella J.F.
CORPORATE SOURCE: Molecular Neurogenetics Unit, Massachusetts General Hospital East, 13th Street,Charlestown, MA 02129, United States.
SOURCE: Human Molecular Genetics, (1993), 2/6 (745-749)
CODEN: HMGEE5 ISSN: 0964-6906
DOCUMENT TYPE: Journal; Article
COUNTRY: United Kingdom
LANGUAGE: English
SUMMARY LANGUAGE: English

AN 1993:23197520 BIOTECHNO

AB **Electrical** excitability of neurons and muscle **cells** is mediated largely through the actions of the voltage-gated sodium channel. Initiation and propagation of the **action potential** is a direct result of the precisely controlled inward flux of sodium through these channels. Much attention has been paid to the sodium channel α-subunit, the major, **pore**-forming component. However, α-subunits are associated with one or more smaller β-subunits, which have been implicated in the critical fine tuning of the gating properties of the channel. To investigate the properties of the β-subunit, we have isolated a cDNA encoding the human brain β1-subunit and assigned the corresponding gene to chromosome 19. We have also examined the effects of expressing the brain β1-subunit on the kinetics of a coexpressed muscle sodium channel α-subunit. Our results underscore the functional importance of the β1-subunit and imply a conserved mechanism for the interaction of the β1-subunit with different α-subunits.

L55 ANSWER 17 OF 19 PASCAL COPYRIGHT 2005 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1993-0523472 PASCAL
TITLE (IN ENGLISH): The cloning and expression of a sodium channel β1-subunit cDNA from human brain
AUTHOR: MCCLATCHEY A. I.; CANNON S. C.; SLAUGENHAUPT S. A.; GUSELLA J. F.

CORPORATE SOURCE: Massachusetts gen. hosp., molecular neurogenetics unit, Charlestown MA 02129, United States
SOURCE: Human molecular genetics, (1993), 2(6), 745-749, 27 refs.
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-22540, 354000033976850210
AN 1993-0523472 PASCAL
AB **Electrical** excitability of neurons and muscle **cells** is mediated largely through the actions of the voltage-gated sodium channel. Initiation and propagation of the **action potential** is a direct result of the precisely controlled inward flux of sodium through these channels. Much attention has been paid to the sodium channel α-subunit, the major, **pore**-forming component. However, α-subunits are associated with one or more smaller β-subunits, which have been implicated in the critical fine tuning of the gating properties of the channel. To investigate the properties of the β-subunit, we have isolated a cDNA encoding the human brain β1-subunit and assigned the corresponding gene to chromosome 19. We have also examined the effects of expressing the brain β1-subunit on the kinetics of a coexpressed muscle sodium channel α-subunit

L55 ANSWER 18 OF 19 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 91:70123 LIFESCI
TITLE: Noninvasive recording of receptor **cell action potentials** and sustained currents from single taste buds maintained in the tongue: The response to mucosal NaCl and amiloride.
AUTHOR: Avenet, P.; Lindemann, B.
CORPORATE SOURCE: Dep. Physiol., Univ. Saarlandes, D-6650 Homburg/Saar, FRG
SOURCE: J. MEMBR. BIOL., (1991) vol. 124, no. 1, pp. 33-41.
DOCUMENT TYPE: Journal
FILE SEGMENT: M; R
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Apical membrane currents were recorded from the taste **pore** of single taste buds maintained in the tongue of the rat, using a novel approach. Under a dissection microscope, the 150- mu m opening of a saline-filled glass pipette was positioned onto single fungiform papillae, while the mucosal surface outside the pipette was kept dry. **Electrical** responses of receptor **cells** to chemical stimuli, delivered from the pipette, were recorded through the pipette while the **cells** remained undamaged in their natural environment. We observed monophasic transient currents of 10-msec duration and 10-100 pA amplitude, apparently driven by **action potentials** arising spontaneously in the receptor **cells**.

L55 ANSWER 19 OF 19 LIFESCI COPYRIGHT 2005 CSA on STN
ACCESSION NUMBER: 82:65756 LIFESCI
TITLE: Ionic channels in skeletal muscle.
AUTHOR: Stefani, E.; Chiarandini, D.J.
CORPORATE SOURCE: Dep. Physiol., Cent. Estudios Avanzados del Inst. Politecnico Nacil., Apartado Postal 14-740, Mexico 14, D.F., Mexico
SOURCE: ANNU. REV. PHYSIOL., (1982) vol. 44, pp. 357-372.
DOCUMENT TYPE: Journal
TREATMENT CODE: General Review
FILE SEGMENT: M
LANGUAGE: English
AB There is now considerable evidence that ions can move across the **cell** membrane through voltage-gated aqueous **pores** celled

"ionic channels." Each channel has a characteristic permeability, selectivity, and kinetics. **Electrical** excitation in skeletal muscle involves voltage- and time-dependent changes of the permeabilities to Na super(+) and K super(+) which induce a transient inflow of Na super(+) into the fiber followed by an outflow of K super(+). As a result of these ionic movements the **action potential** is generated. Besides these Na super(+) and K super(+) channels, a voltage-dependent Ca super(2+) channel has been described recently in frog skeletal muscle. At rest, Cl super(-) and other K super(+) channels are responsible for the dominant conductance. Most of these K super(+) channels rectify inward current. Here The authors review recent research on ionic channels in twitch and tonic skeletal widely used.